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Effects of Indoor Air Pollution on the Development of Children under Five Years of Age in Sri Lanka

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Abstract: Air pollution is a multifaceted environmental toxin affecting the Central Nervous System (CNS) through diverse pathways. The CNS of young children is particularly susceptible to the detrimental effects of toxins, as brain development continues postnatally with the formation of interneuronal connections, glial cell proliferation and myelination of axons. Indoor air pollution (IAP) from solid fuel combustion is more harmful than outdoor air pollution. Numerous air pollutants hazardous to health are released during the burning of unprocessed biomass. The primary source of fuel in Sri Lanka for cooking is biomass, mainly wood. In this study, we evaluated the influence of IAP resulting from biomass combustion on the neurodevelopment of children. In a cohort of children under five years living in a semi-urban area of Sri Lanka, neurodevelopment was assessed using Denver II developmental screening test. Air quality levels were measured (Carbon Monoxide (CO) and Particulate Matter 2.5 ($PM_{2.5}$)) in a subsample. There were significantly high levels of CO and $PM_{2.5}$ in the ambient air of households using biomass as the primary fuel for cooking. Children living in these households had a significantly higher number of children with 'suspect' developmental assessment scores in the language, social behavior and play and gross motor development domains.

Keywords: indoor air; language development; gross motor development; carbon monoxide; particulate matter 2.5; solid fuel combustion

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1. Introduction

Indoor air pollution (IAP) from solid fuel combustion is an important risk factor for ill health amongst children [1]. A large number of air pollutants are released during the burning of unprocessed biomass (wood, animal dung, crop residues, and grasses) for cooking and space heating. Despite its ill effects on health, the importance of the reduction of IAP has not received due attention. Unprocessed solid fuels remain an important source of fuel in developing countries in Southeast Asia and sub-Saharan Africa [2]. Biomass fuels are at the lower end in terms of combustion efficiency and cleanliness. Hazardous pollutants in the smoke emitted from stoves using biomass fuel include respirable particulate matter, carbon monoxide (CO), nitrogen oxides, formaldehyde, benzene, 1,3 butadiene, polycyclic aromatic hydrocarbons (such as benzo[a]pyrene), and many other toxic organic compounds [3].

A stove without a chimney produces very high concentrations of air pollutants even when compared with the worst outdoor settings. Open stove combustion of biomass fuel,

consisting mainly of solids, produces high concentrations of particulate matter ranging between 2000 and 15,000 mg/m^3 [4,5].

The central nervous system (CNS) of young children is particularly susceptible to detrimental effects of toxins because brain development continues postnatally with the formation of intraneuronal connections, glial cell proliferation and myelination of axons [6]. The maturation of the cortex during the first years of life is very intensive, and the frontal cortex is the last to mature [7]. This period of life is considered an important window for brain development since the brain's plasticity decreases with age. Due to the long period of the vulnerable developmental process, susceptibility to environmental insults is high [8].

In a study on autopsies of children and young adults resident in Mexico, ultrafine particles were found to accumulate in the nasal epithelium, olfactory neurons and in the endothelium and basement membranes of olfactory bulb arterioles, together with immunoreactivity to beta-amyloid 42 and alpha-synuclein [9]. In humans, exposure to severe air pollution is associated with increased levels of an inflammatory mediator cyclooxygenase-2, and accumulation of the 42-amino-acid form of b-amyloid, which is a cause of neuronal dysfunction [10].

Few studies have assessed the effect of indoor air pollution on early childhood neurodevelopment of humans; these studies show a negative relationship between IAP and neurodevelopment during early childhood [11,12]. These studies have concluded that more research is needed to ascertain the relationship between IAP and neurodevelopment. There is a paucity of data on the effects of indoor air pollution due to solid fuel combustion on the neurodevelopment of pre-school-age children. In this study, we investigated the association between IAP due to solid fuel combustion and neurodevelopment of children under five years of age in a semi-urban area in Sri Lanka. The findings of this study will benefit the scientific community to develop new guidelines to prevent the possible hazardous exposures of young children to indoor air pollution.

2. Materials and Methods

2.1. Study Setting

This prospective study was conducted in 2014 within the Ragama Medical Officer of Health (MOH) area, situated in the Gampaha District of Sri Lanka which is one of the most populous districts of the country with an estimated population of 2.3 million [13]. Gampaha, specially Ragama MOH area, has urban, semi-urban and rural characteristics and a multiethnic population. According to the 2012 census, approximately 63% of households in the Ragama MOH area used biomass fuel and 31% used LP gas [13].

2.2. Recruitment of Children

A total of 262 children who permanently reside in the area were recruited. This study was an ancillary study of a birth outcome study conducted to assess the effects of air pollution on fetal life and the immediate postpartum period. Children living in households where air quality was measured were recruited. Children with any congenital abnormality, birth trauma or any history of birth asphyxia, physician-diagnosed chronic illness or syndromic disease, with documented immunodeficiency or diagnosed to have any chronic disease were excluded.

2.3. Baseline Assessment

Socioeconomic background and sources of cooking fuels information of the household were gathered by using an interviewer-administered questionnaire. The initial interview of the mother to obtain consent and to get an appointment to visit the households to measure air quality levels was performed in the well-baby clinic setting while they were waiting for the vaccinations or for weight measurements. The detailed interview was conducted after visiting the households to get the air quality measurements and to assess the development of the child. The mothers or guardians of the children were interviewed. The "High exposure group" was defined as the children residing in households where firewood or

kerosene oil is used as the main energy source for cooking. The "Low exposure group" was comprised of the children from households using LP gas or electricity as the main energy source. Thereby the exposure status was categorized according to the major fuel type used for cooking.

2.4. Developmental Assessment

The Denver II developmental screening test was used to assess the neurodevelopment of children. A medical doctor was trained to do the assessment. Neurodevelopment assessment pertaining to all four major domains of development was carried out. The Denver II developmental screening test comprising of 125 test items/tasks assesses neurodevelopment of children under four domains: social behavior and play, fine motor, language and gross motor. The Denver II developmental screening test is used to screen children under six years of age [14]. A neurodevelopmental assessment of all four domains was carried out.

The social behavior and play domains assess behavioral adaptation of the child to get along with people and society. The fine motor domain assesses the ability of a child to manipulate small objects, hand-eye coordination and problem solving. The language domain assesses the use of the language, hearing and understanding ability of the language of a child in his or her native language. The gross motor domain assesses major motor activities such as walking, running and other gross motor activities needed for independent survival.

Each item was assessed according to the instruction given in the training manual of the Denver II developmental screening test. Items to be tested were selected according to the chronological age of the child. The recommended correction for age was performed for premature children. 'Development delay' was defined as 'a child not performing a task which could be performed by all normal children (100%) of the same age'. 'Cautions for development delay' were defined as 'a child not being able to perform an item/task, which was performed by75% of the normal children of the same age'. Data were recorded in the standard Denver II catalog. A standard Denver II test kit was used to administer the test items. According to the Denver II scoring system, a child is labeled as a "suspect" for a particular developmental domain if the child had two or more cautions or one or more delays. If not, the child was labeled as "normal" for that developmental domain at the time of the assessment. Children were recruited to the study from the child welfare clinics conducted in their residence area. Informed written consent was obtained from the parents or the guardian of the child before recruitment. Once the child was recruited, the initial baseline questionnaire was filled out at the same time and an appointment was made with the parents according to their free time to visit their home and to do the air quality measurement and to assess the development of the child. All the developmental assessment tests were conducted at the child's home spending about 30 to 60 min with the child depending on the cooperation and behavior pattern of the child. The picture of the horse in the language domain was replaced with a cow as the horses are not seen in Sri Lanka. Children of both "high" and "low" exposure groups were scored as suspect or normal for each domain. Data were entered into EPIDATA databases (separately for each source of data) and analyzed using SPSS version 16 software.

2.5. Air Quality Assessment

Air quality measurements were performed in a subsample of households. Air pollutant levels in the kitchen during cooking hours were recorded using air quality measuring equipment. Concentrations of particulate matter 2.5 ($PM_{2.5}$) and CO were measured for two consecutive hours with minute-to-minute recording during the preparation of the lunch meal using two real-time monitors. A DustTrak II monitor (DUSTTRAK™, TSI, Shoreview, MN, USA) was used to measure $PM_{2.5}$ and a TSI's Q-Trak monitor (Shoreview, MN, USA) was used to measure CO_2 and CO [15].

2.6. Data Analysis

Descriptive statistics were used to describe the sociodemographic profile of the cohort. Differences between the high and low exposure groups were compared using the chi-square test. Odds ratios and their 95% confidence intervals were calculated for the presence of developmental concerns for all four domains separately. Logistic regression analysis was used to control for confounding factors associated with early life development.

3. Results

The "high" exposure group comprised 59% of recruited children. Maternal education, paternal education, family income, having a chimney, cooking duration and having a sibling were significantly different between the two exposure groups [15].

A significantly higher proportion of children in the high exposure group were assessed as "suspects" in the language developmental domain (OR = 2.34, 95% CI—1.32–4.13), in the social behavior and play domain (OR = 1.86, 95% CI—1.09–3.16) and in the gross motor domain (OR = 2.50, 95% CI—1.28–4.89) as compared to children in the low exposure group (Table 1).

Logistic regression analysis was conducted using the language development domain, gross motor development domain, social behavior and play as the dependent variables and exposure status, parental education, family income, age and the child having a sibling as independent variables. The odds of a child being classified as a "suspect" in the language development domain was significantly higher in children in the high exposure group (a OR = 2.162; 95% CI: 1.185–3.941) as compared to children in the low exposure group, after adjusting for above-mentioned confounding variables (Table 2). Children under three years (a OR = 0.475; 95% CI: 0.271–0.835) were significantly less likely to be classified as a "suspect" as compared to children >three years in the language domain after controlling for other variables (Table 2). The odds of a child being classified as "suspect" in the social behavior and play domain was significantly higher in children in the high exposure group (a OR = 1.773; 95% CI: 1.022–3.077) as compared to children in the low exposure group (Table 2). In the gross motor development domain, a child being identified as "suspect" was significantly higher in children in the high exposure group (a OR = 2.102; 95% CI: 1.046–4.225) and in offspring of less-educated fathers (a OR = 2.933; 95% CI: 1.239–6.943) after adjusting for the other variables (Table 2).

Having a delay in one developmental domain showed a significantly higher possibility of having problems with achieving developmental goals in the rest of the domains also (Table 3). It is much stronger in the fine motor and language domains (Table 3).

Carbon monoxide, carbon dioxide and particulate matter 2.5 levels were measured in 115 households; particulate matter 2.5 and carbon monoxide were significantly higher in the high exposure group households [15]. The available data on outdoor air pollution in the nearest city (Gampaha) suggests that the levels of particulate matter it is lower than that inside kitchens during cooking in the "high" exposure group. The mean carbon monoxide levels outdoors were lower than indoor levels in kitchens during cooking in both high and low exposure households. [16].

Table 1. Development assessment by selected socio-demographic characteristics.

Characteristic	Development Domain											
	Social Behavior and Play Domain			Language Domain			Fine Motor Domain			Gross Motor Domain		
	Suspects N (%)	Normal N (%)	OR (95% CI)	Suspects N (%)	Normal N (%)	OR (95% CI)	Suspects N (%)	Normal N (%)	OR (95% CI)	Suspects N (%)	Normal N (%)	OR (95% CI)
Group												
High exposure [1] (*n* = 145)	63 (43.4)	82 (56.6)	1.859	57 (39.3)	88 (60.7)	2.337	41 (28.3)	104 (71.7)	1.596	40 (27.6)	105 (72.4)	2.503
Low exposure [2] (*n* = 106)	31 (29.2)	75 (70.8)	(1.092–3.164)	23 (21.7)	83 (78.3)	(1.323–4.131)	21 (19.8)	85 (80.2)	(0.88–2.91)	14 (13.2)	92 (86.8)	(1.28–4.89)
SexMale (*n* = 134)	57 (42.5)	77 (57.5)	1.601	38 (28.4)	96 (71.6)	0.707	35 (26.1)	99 (73.9)	1.178	25 (18.7)	109 (81.3)	0.696
Female (*n* = 117)	37 (31.6)	80 (68.4)	(0.95–2.69)	42 (35.9)	75 (64.1)	(0.42–1.20)	27 (23.1)	90 (76.9)	(0.66–2.10)	29 (24.8)	88 (75.2)	(0.380–1.273)
Age 3 years (*n* = 138)	46 (33.3)	92 (66.7)	0.649	34 (24.6)	104 (75.4)	0.48	37 (26.8)	101 (73.2)	1.282	31 (22.5)	107 (77.5)	1.133
≥3years (*n* = 108)	47 (43.5)	61 (56.5)	(0.39–1.09)	44 (40.7)	64 (59.3)	(0.28–0.82)	24 (22.2)	84 (77.8)	(0.71–2.31)	22 (20.4)	86 (79.6)	(1.06–2.10)
Father's education												
Up to O/L [3] (*n* = 167)	64 (38.3)	103 (61.7)	1.118	59 (35.3)	108 (64.7)	1.639	43 (25.7)	124 (74.3)	1.186	45 (26.9)	122 (73.1)	3.07
Above O/L (*n* = 84)	30 (35.7)	54 (64.3)	(0.65–1.93)	21 (25.0)	63 (75.0)	(0.91–2.95)	19 (22.6)	65 (77.4)	(0.64–2.20)	9 (10.7)	75 (89.3)	(1.42–6.65)
Mother's education												
Up to O/L (*n* = 154)	58 (37.7)	96 (62.3)	1.024	58 (37.7)	96 (62.3)	1.024	42 (27.3)	112 (72.7)	1.444	37 (24.0)	117 (76.0)	1.488
Above O/L (*n* = 97)	36 (37.1)	61 (62.9)	(0.61–1.73)	36 (37.1)	61 (62.9)	(0.61–1.73)	20 (20.6)	77 (79.4)	(0.79–2.65)	17 (17.5)	80 (82.5)	(0.78–2.83)
Income												
Up to SLR [4] 20,000 (*n* = 55)	20 (36.4)	35 (63.6)	0.942	17 (30.9)	38 (22.2)	0.944	12 (21.8)	43 (78.2)	0.815	16 (29.1)	39 (70.9)	1.706
> SLR 20,000 (*n* = 196)	74 (37.8)	122 (62.2)	(0.51–1.75)	63 (32.1)	133 (67.9)	(0.50–1.80)	50 (25.5)	146 (74.5)	(0.40–1.67)	38 (19.4)	158 (80.6)	(0.86–3.37)
Having an industry causing air pollution												
Yes (*n* = 100)	19 (40.4)	28 (59.6)	1.167	16 (34.0)	31 (66.0)	1.129	16 (34.0)	31 (66.0)	1.773	18 (38.3)	29 (61.7)	2.897
No (*n* = 117)	75 (36.8)	129 (63.2)	(0.61–2.23)	64 (31.4)	140 (68.6)	(0.58–2.21)	46 (22.5)	158 (77.5)	(0.89–3.52)	36 (17.6)	168 (82.4)	(1.45–5.77)

[1] High exposure group refers to children living in households using biomass as the major type of cooking fuel, [2] Low exposure group refers to children living in households using LP gas or electricity as the major type of cooking fuel, [3] O/L refers to General Certificate of Education Ordinary Level (11 years of formal schooling): [4] SLR refers to Sri Lankan Rupees (1 USD ≈ 130 SLR at the time of the study).

Table 2. Summary of logistic regression analysis for language development assessment.

Variable	Language Domain		Social Behavior and Play Domain		Gross Motor Domain	
	Significance	Odds Ratio (95% CI)	Significance	Odds Ratio (95% CI)	Significance	Odds Ratio (95% CI)
Constant	0.930	1.089	0.561	1.696	0.049	0.122
High exposure [1]	0.012	2.162 (1.185–3.941)	0.042	1.773 (1.022–3.077)	0.037	2.102 (1.046–4.225)
Father's education (up to O/L) [2]	0.392	1.348 (0.680–2.674)	0.760	1.102 (0.590–2.058)	0.014	2.933 (1.239–6.943)
Mother'seducation(up to O/L) [3]	0.293	1.430 (0.734–2.783)	0.666	0.873 (0.463–1.614)	0.488	0.765 (0.358–1.632)
Family income (<SLR 20,000) [4]	0.409	0.746 (0.372–1.495)	0.731	0.891 (0.463–1.718)	0.206	1.607 (0.771–3.351)
Age (< 3 years) [5]	0.010	0.475 (0.271–0.835)	0.114	0.654 (0.386–1.108)	0.546	1.218 (0.642–2.310)
Having a sibling [6]	0.403	1.282 (0.716–2.294)	0.908	1.033 (0.601–1.775)	0.568	1.212 (0.626–2.346)

[1] Reference group is low exposure group using LPG and electricity for cooking, [2] Reference group is father's education above ordinary level, [3] Reference group is mother's education above ordinary level, [4] Reference group is having an income of SLR 20,000 (1 USD ≈ 130 SLR at the time of the study) or more, [5] Reference group is the children aged three years or more at the recruitment, [6] Reference group is children not having siblings.

Table 3. Correlation between scores of different development domains.

Developmental Domain	Correlation Coefficient	Developmental Domain			
		Social	Language	Fine Motor	Gross Motor
Social development delays score	Pearson Correlation	1			
	Sig. (2-tailed)				
	N	222			
Language development delays score	Pearson Correlation	0.219 **	1		
	Sig. (2-tailed)	0.001			
	N	222	222		
Fine motor development delays score	Pearson Correlation	0.187 **	0.503 **	1	
	Sig. (2-tailed)	0.005	<0.001		
	N	222	222	222	
Gross motor development delays score	Pearson Correlation	0.182 **	0.486 **	0.399 **	1
	Sig. (2-tailed)	0.007	<0.001	<0.001	
	N	222	222	222	222

** significance at $p < 0.01$.

4. Discussion

The influence of environmental factors on the neurodevelopment of children is well established. There is growing evidence to suggest that indoor air pollution adversely affects the neurodevelopment of children. However, the evidence in the medical literature is limited and inconclusive. Thus, this study was conducted by us in the Ragama MOH area to study the effects of IAP on the neurodevelopment of children. Based on our air quality levels measurements and in the published literature [17–19], we considered children living in households mainly using firewood or kerosene oil for cooking as the "high" exposure group and children living in households mainly using LP gas or electricity as the "low" exposure group. Our study revealed a negative influence of IAP on gross motor, social behavior and play, and language domains of neurodevelopment of children.

The neurodevelopment of a child depends on neuronal plasticity, which permits the nervous system to reorganize its neuronal network in response to stimulations from the

environment. This environmental influence on neurodevelopment could be either positive or negative. Acquiring a developmental milestone involves learning, remembering and finally developing the skills required for that task. Thus, the environment a child is brought up in will directly influence neurodevelopment [20,21]. In this study, we found that three out of four main neurodevelopmental domains are affected in children living in an environment having high indoor air pollution levels. Golding et al. stated that at least half of the variance in motor development among children is likely to be influenced by the environment. Many factors during pregnancy are implicated with adverse neurodevelopmental outcomes. These include: alcohol consumption, diabetes, use of antidepressant drugs, iodine or iron deficiency and lack of dietary fish intake. Low birth weight and maternal postnatal depression have also been implicated with adverse developmental outcomes [22].

In an adult brain, there are around 100 billion neurons. By three years of age, each neuron develops on average 15,000 synapses [23]. Thus, the human brain undergoes many functional changes during the early years of life, which will determine the final functional capacity of the brain. During functional development of the CNS, frequently used neuronal pathways are enhanced, while unused pathways undergo atrophy. Thus, influences from the environment play a major role in neurodevelopment during early childhood [24]. Numerous toxic substances contained in IAP could adversely affect the normal functional maturation of the brain.

Children in the high exposure group and being older were significant independent risk factors for slower development, after controlling for other confounding factors. Milojevic et al. concluded that exposure to air pollution is time-dependent and, with longer exposure, the effect is stronger; further, cognitive development in children under three years is not significantly affected until about five and seven years of age [25]. This suggests that if the exposure is longer, the effects can be much stronger.

Gross motor development was significantly affected in children in the high exposure group households and offspring of less-educated fathers after adjusting for confounders. There is limited literature on the influence of air pollution on gross motor development. Hernández-Martínez has reported that paternal perception about development is associated with the gross motor development of their children [26].

We found that there was a significant influence of exposure to IAP on development in the social behavior and play domain after adjusting for potential confounding factors. Elizabeth et al. concluded that traffic-related air pollution has a significant influence on language and social development in early life; it may be due to an effect on neurodevelopment in early life [27]. The association between air pollution and social behavior and play is discussed in a review article by Chelsea, recommending further research in the field [28].

There is growing evidence that the neurodevelopmental effects are mainly due to the deposition of particulate matter in the brain and induction of an inflammatory response by the activation of pro-inflammatory cells. It was found that air pollutants can reach the brain via many pathways and that there is a possibility of these pollutants crossing the blood-brain barrier causing adverse effects on neurodevelopment in exposed children [29,30].

Compared to older children and adults, preschool children are more liable to be affected by indoor air pollution, because they spend most of their time indoors. Small children often prefer to stay close to their mother, and thus, are more likely to be in the kitchen during cooking hours. Pregnant females also get exposed to the toxic effects of IAP, which may influence the development of the fetal brain. Prenatal exposure to IAP could be a contributing factor for adverse neurological outcomes observed in children living in households with high IAP.

Studies have revealed that ultrafine and fine particles can penetrate pulmonary tissue and enter the capillaries, reaching organs such as the liver, spleen, kidneys, heart, and brain. Recently, Elder et al. demonstrated that ultrafine particles reach the brain, through the circulation or directly via olfactory nerves [31]. In rats, intra-trachealy instilled radioactive particles of less than 100 nm were subsequently detected in several organs, including the brain [32]. Ultrafine carbon particles [33] and Manganese (Mn) nanoparticles [31] have

been found in the olfactory bulbs, cerebrum, and cerebellum after inhalation. Another possible mechanism of depositing particulate matter (PM) > 200 nm (TiO2) in the brain is through macrophages and dendritic cells [29]. Changes in cytokine expression in brains of mice have been directly linked to intranasal exposure to ultrafine carbon [34]. A recent study conducted by Calabro et al. has concluded that one mechanism for possible brain damage due to air pollution is oxidative stress and impaired mitochondrial function [35].

Neurodevelopment is influenced by many factors such as nutrition, infections, parental education, employment status of the mother, amount of stimulation, etc. [36]. Though children from higher socio-economic backgrounds are likely to get better nutrition and fewer infections, they may get less stimulation if the mother is employed and has less time to spend with the child. Thus, confounding factors that would influence the findings of our study need to be excluded. Even after adjusting for confounding factors, high IAP had a negative impact on the neurodevelopment of pre-school children in the language, social and play, and gross motor domains. However, further studies are required to establish this fact for certain.

The main limitation of this study is that we could not assess some contributing factors such as nutrition in this cohort of children as it may significantly vary even during the course of a week. More studies with larger samples are needed to confirm our findings.

5. Conclusions

High levels of air pollutants affected pre-school children's neurodevelopment in the language, gross motor and social behavior and play domains. The longer the duration of exposure, the greater the effect on language development.

Author Contributions: N.R. did the data collection, air quality measuring, data entering, data analyzing and writing the manuscript. R.W., P.P. and N.S. analyzed and interpreted the data and were involved in manuscript writing. S.N. and A.K. were involved in data collection and manuscript writing. All authors have read and agreed to the published version of the manuscript.

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Institutional Review Board Statement: Ethical clearance was obtained from the Ethical Review Committee of the Faculty of Medicine, University of Kelaniya (P025/04/2011).

Informed Consent Statement: The nature and process of the study was explained to the parents/guardians of selected children. Written informed consent was obtained prior to recruitment. Confidentiality of the information was ensured. Children requiring specialized care were referred to consultants at the Colombo North Teaching Hospital. All mothers living in households with high IAP were advised how to mitigate IAP.

Data Availability Statement: All the data and the references are given in the manuscript itself.

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